A SECOND CONTRIBUTION TO THE STUDY OF LOCALIZED CEREBRAL LESIONS.*

By E. C. SEGUIN, M. D.

IN 1877 I reported to the American Neurological Association[1] a number of cases with accurate *post-mortem* examinations, illustrating the doctrine of localization of functions in the brain. Since that time I have made several similar observations, some of which have been published as isolated cases. In the past year two remarkable cases of cerebral tumor bearing upon the Ferrier hypothesis have been added to my records, and I think that the time has come to offer a second instalment of facts in this department of medicine to the medical public. I shall first relate my last unpublished cases, and point out their significance, then reproduce in brief the isolated observations, positive and negative, which I have separately published.

I would only claim, in offering this second paper, to be adding a few data, trustworthy data, I believe, to a mass of observations which tend to support the theory of cerebral localization. This theory or hypothesis can be established as true only by great numbers of pathological facts corroborating the results of experimental physiology and of anatomy.

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* Read by title at the seventh annual meeting of the American Neurological Association, June 17, 1881.

[1] Contribution to the study of localized cerebral lesions. *Transactions of the American Neurological Association*, vol. ii, 1877.

CASE I.—Mrs. I. D., aged 58 years, seen Oct. 3d, 1880.

A strong, intellectual woman, who has enjoyed good health. In early spring was overworked and anxious about the outfit of a daughter who was to be married.

In May began to have a peculiar general headache (different from any she had had before), most marked in the occipital region, and always worst at night. She often complained of a sore, stiff feeling in the neck on rising in the morning. At times, in connection with headache, has had nausea and vomiting. This headache has been a prominent symptom ever since, amounting at times to agony.

Later in the month of May, or in the early part of June, there was noticed a trembling of the left hand; this increased, and was accompanied by evident loss of power. Relatives of the patient describe two sorts of movements of the left arm: first, a slight and nearly constant fine tremor; and, second, attacks of considerable jerking, so that the patient was obliged to hold the affected left hand with the right. Each day there were several such attacks, some lasting an hour.

Has grown steadily worse; more headache, marked paresis of the left arm, with some contracture, slight weakness of the left leg. Sight not so good as formerly, but there has been no diplopia, hemiopia, etc.

Last night the pain was intense through the mastoid regions, and in the whole of the head. Was given $\frac{1}{8}$ grain sulphate of morphia occasionally, and by 10 A. M. to-day had taken $\frac{5}{8}$ grain; is semi-comatose, but still groaning from pain; the left hand and arm are semi-flexed and stiff.

Examination at 5 P. M. Patient is profoundly asleep, yet can be roused; respiration is slow and very irregular, but not of the Cheyne-Stokes type. When spoken to loudly, points (with right hand) to the sides of the head as the seat of chief pain; is able to swallow. The pupils are small and fixed, the right larger. The right internal rectus is weak. The left lower face is paretic. The left arm and hand are strongly adducted and semi-flexed on the thorax, and passive extension is difficult and painful. Legs extended, not stiff; both show good reflexes at the knees. Left hand and leg are less sensitive than the right. The pulse beats about 72 per minute, and is weak; the axillary temperature is 37.4° C. (99.3° F.). After the use of atropia, I was able to observe typical neuro-retinitis (choked disks) in both eyes; no hemorrhages. Urine contains a trace of albumen.

My diagnosis was tumor in the right cerebral hemisphere, complicated by morphia narcosis. I considered that very probably the tumor was in the median region of the hemisphere, in the so-called centres for the arm and leg, according to Ferrier's experiments and to recent *post-mortem* facts.

A great many notes were made during the progress of the case, but they only show the extraordinary variations in the state of the patient, which I, and others, have observed in cases of cerebral tumor. Some days Mrs. D. would be sitting up and very bright, and the next day might appear moribund.

On October 5th is up on a lounge, is bright and cheerful, though mind wanders at times; headache has returned about the vortex. Can converge eyes well. Exhibits common left hemiparesis, with contracture, most marked in arm and hand. Ordered solid food, and iodide of potassium.

Oct. 10th. Growing steadily worse. Attacks of pain in the head, at times very severe, controlled by morphia and chloral. The arm is now completely paralyzed, with painful contracture of elbow and shoulder. No voluntary motion in left arm for forty-eight hours; the left leg, which four days ago could be drawn up fairly well, is now nearly motionless. Left face is paretic, but tongue points straight. Answers questions, but wanders; wants to be dressed, to go out, etc. Wets the bed. Optic nerves choked as before.

Oct. 13th. State of paralyzed limbs has varied from partial to complete paralysis. Extreme sensibility to narcotics.

Oct. 15th. Sulphate of quinia produced delirium the other evening, and she is easily plunged into dangerous narcosis by morphia. Morphia .002 + and chloral .15 have *some* effect.

[On Oct. 9 it is noted that left arm is completely relaxed and the tongue is straight.]

Nov. 1st. Divergent strabismus and slight drooping of right upper lid. Speech very indistinct. Left hemiplegia as above. Delirious and semi-comatose at different times. Incontinence of urine and fæces.

Nov. 4th. Greater coma and first appearance of fever. 7.30 A.M.: Pulse, 162; respiration, 52. At 4.30 P.M., pulse, 136; axillary temperature, 39.2° C. (102.5° F.); breathing, moribund *i. e.*, inspiration and expiration equal. Left arm in semi-flexion on chest, elbow and wrist limber, fingers slightly but decidedly contractured. At 10 P.M., respiration, 56; pulse, 160; axillary temperature (six minutes), 39.8° C. (103.75° F.). Right eye is in slight external

strabismus and motionless ; the left is in continual lateral motion ; pupils medium-sized, equal.

Nov. 5th, 1 A.M. Respiration, 56 ; pulse, 176 ; axillary temperature, 40.15° C. (104.25° F.) ; jaws firmly closed. Death occurred before daylight, and the temperature finally rose to 40.6° C. (105° F.).

No *post-mortem* measurements could be made.

The autopsy was made about ten hours after death by Dr. R. W. Amidon under my direction. Drs. W. R. Birdsall and C. Adam were also present.

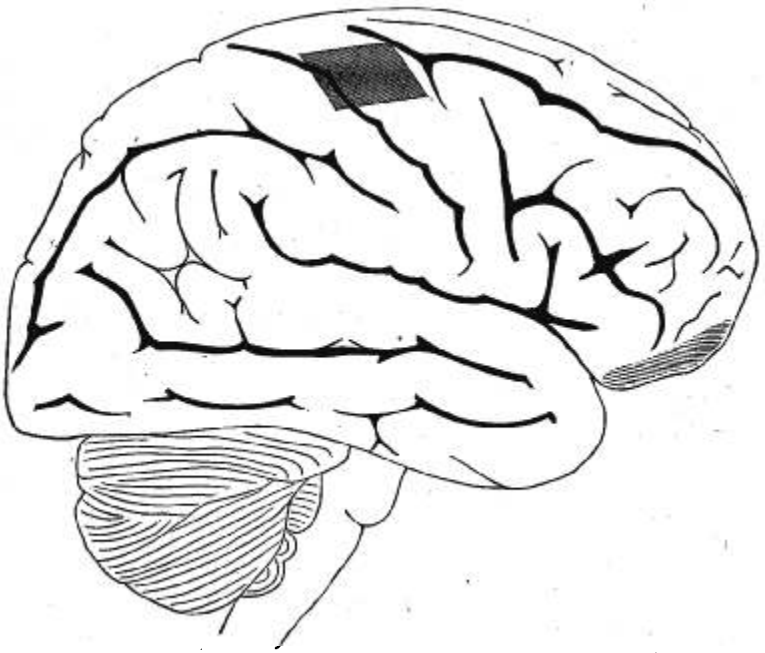

FIG. 1.

Lateral view of the right cerebral hemisphere, after Ecker. Shaded spot represents the location of the tumor. Superficially it involved only the ascending frontal gyrus.

Very little blood escaped on removing the calvarium. The pia mater was found excessively dry and sticky and without gloss. There was a marked prominence of the right parietal portion of the brain, causing the whole hemisphere

to appear much larger than the left. The convolutions about the upper end of the fissure of Rolando on the right side were very much flattened.

A vertical transverse section passing through the middle of the motor zone revealed a consistent, grayish-red

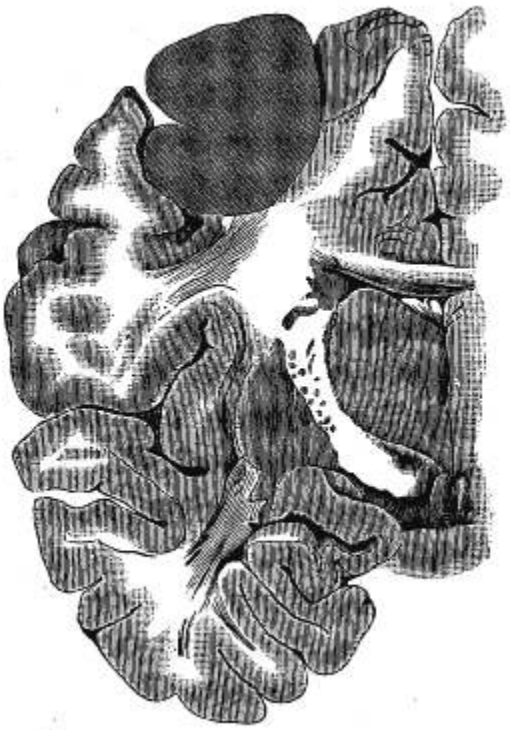

FIG. 2.

Transverse vertical section through the right hemisphere, anterior view; after photo. No. 5 of Bitot. The gray shaded mass in the upper part of the figure represents the tumor.

tumor lying chiefly in the right ascending frontal convolution, wholly under the pia, and in the angle formed by the ascending frontal convolution and the paracentral lobule at the top of the brain. See fig. 1.

The tumor was about the size of a small English walnut,

well defined from the brain substance, vascular, and at points almost gelatinous in structure.

The right third nerve was grayish. Right eye removed, showed an elevated papilla.

The brain and eye were placed in bichromate of potassium solution for hardening.

The following is a study of the topography of the lesion made upon the hardened specimens:

The tumor, ovoid in shape, lies in the upper part of the ascending frontal convolution and in its subjacent white matter. It measures upon the vertical transverse section of the brain, transversely, 15 mm. at its pia mater attachment, 20 mm. in its middle, and, vertically, from its deepest point to the pia 28 mm. See fig. 2.

It extended well across the bottom of the fissure of Rolando, so as to slightly impinge upon the ascending parietal gyrus. The distance from the surface of the brain in the longitudinal fissure to the internal edge of the tumor is 25 mm., thus leaving the paracentral lobule and its attached white matter intact.

The tumor is spongy in texture, well defined from the surrounding cerebral substance, and seems firmly united to the pia. The microscope shows it to be an alveolar carcinoma.

It probably caused a great deal of pressure in spite of its small size.

CASE 2—L. K., an upholsterer, aged 34 years, came to the Manhattan Eye and Ear Hospital, department for nervous diseases, Oct. 6, 1879. He was a strong and healthy-looking German. The following is a transcript of my notes:

Has had attacks of right-sided epilepsy. First seizure was about two years ago (1877), and the attacks have occurred at the rate of one every four or six weeks. In the last few months has had attacks every week, and even several times a week. The phenomena have always been the same in these numerous attacks;

the spasms being wholly restricted to the right arm and leg; the slightest attacks are only momentary shocks on the right side of the body—no spasm in the face. Even in the severe attacks the spasm is wholly clonic, and he never loses consciousness. An exception to this occurred on August 5, 1879, when he had a severe seizure with loss of consciousness.

The attacks last from a few seconds to a few minutes; they are preceded by a sensation of something rising from below upward to the throat, and there causing choking. He never foams at the mouth, or bites tongue, or micturates in attacks, and during them he is often able to speak a few words in a jerky manner.

In intervals between attacks has good use of his right hand and leg; he is now working at his trade. Mind clear and calm.

Very lately has noticed a slight weakness in the right limbs, and the right leg has been the seat of an indefinite numbness. Complains of diffused headache, mostly frontal. No vertigo or petit-mal.

Denies injury to head and any venereal disease.

Examination.—Manner, appearance, and speech normal. No facial palsy; tongue straight; pupils equal. Right hand grasps 45° and 48°, and the left 45° and 45° on Mathieu's dynamometer. No anæsthesia to careful testings. Patellar tendon reflex absent on the left side, and strong on the right (never sharp pains in legs). The walk is rather of hemiplegic type on the right side; the right foot is held slightly in equino-varus position. Complains of sight of right eye, and states that when a soldier he was obliged to aim with the left eye. Examination of eyes by Dr. J. O. Tansley shows myopia of right eye, but optic nerves normal.

The diagnosis was a cortical lesion (tumor?) in the left hemisphere, involving the upper part of the motor area.

The following mixture was ordered: ℞. potassii iodidi, 15.; potassii bromidi, 30.; aquæ, 200.; S.: one teaspoonful before each meal, and two at bedtime, in plenty of water.

Oct. 10th. No spasm since beginning of the treatment, but the paralytic phenomena have increased; the walk is distinctly hemiplegic on the right side. Still works. Ordered to continue treatment, with addition of 4. ext. ergotæ fld. with the evening dose of bromide.

Oct. 13th. No attack. Speech normal; tongue deviates slightly to the right.

Oct. 17th. Slight spasm in the arm (right) yesterday; increasing paresis. Right hand squeezes 44° and 45°; the left, 50° and

45°. Ordered only three teaspoonfuls of bromide mixture at bed-time. To take besides 20 drops of a saturated solution of iodide of potassium three times a day in water.

Nov. 10th. No spasm; paresis of right leg more marked; walk distinctly hemiplegic.

Nov. 20th. Dr. Amidon was summoned to see the patient at his house. Has violent headache, more to the left of the median line at the vertex; photophobia, nausea, and almost constant vomiting. There is complete paralysis of the right arm and leg, and these parts are œdematous. Partial relief by hypodermic injection of .02 sulphate of morphia thrice during the day.

Nov. 22d. The pain has continued intense. Has asked to be killed. No aphasia. Eyes, examined by ophthalmoscope, show myopia $\frac{1}{10}$ in each eye; fundus normal; sleep induced by hypodermic injection of chloral.

Nov. 30th. Headache has continued intense, requiring chloral and morphia. Has also had bromide and iodide of potassium as above. Some motion in fingers and right foot (lost on 31st).

Nov. 14th. Less headache, but continued right hemiplegia. Bed-sore beginning over sacrum. Some hesitancy of speech. At no time any aphasic defect.

Nov. 19th. Eyes again examined (without atropine); right fundus well seen, and found normal.

Nov. 21st. First signs of paresis in face; right cheek looks weak, and tongue points a little to the right side. Still has very severe headache.

Nov. 30th. Involuntary escape of urine. Scarcely able to speak from difficulty of articulation. At times silly.

Dec. 4th. Cannot be understood. Some contracture at right elbow, and the muscles of right arm and leg show some atrophy.

Dec. 19th. Paralysis now very marked about right cheek.

Dec. 31st. Quite a large bed-sore has formed on the right side of the sacrum. Marked atrophy of right arm and leg; elbow very stiff. Is semi-comatose. Pupils moderately small. Understands what is said to him, and tries to protrude his tongue when asked. Profuse sweating.

Jan. 2, 1880. Much brighter; speech can be understood. Of late has had no treatment except chloral occasionally.

Jan. 4th. Beginning of terminal stage. Fever and rapid respiration. A.M., axilla temperature, 38.8° C. (101.80° F.). At 5 P.M., asleep and sweating profusely. Pulse, 126; respiration, 26;

temperature, 39.2° C. (102.5° F.) in axilla; in the rectum the thermometer indicates 40.1° C. (104.12° F.).

Jan. 5th. Fever and rapid respiration all night. At 11 A.M., pulse, 126; respiration, 56 (shallow); rectal temperature, 41.25° C. (106.2° F.). At 2 P.M., comatose without stertor; skin moist. Eyes in conjugate deviation to the right side; head straight. Pulse, 145; respiration, 50; rectal temperature, 41.6° C. (106.8° F.). At 4 P.M., died.

The autopsy was made by Drs. R. W. Amidon and W. R. Birdsall 24 hours after death. The calvarium was found very thin; translucent in spots. Dura mater normal. No subarachnoid fluid. There were many large superficial cerebral veins. The left motor area gave a sense of fluctuation; the convolutions of this part seemed normal, but were flat. On attempting to remove the falx cerebri in the usual manner, it was found adherent to the inner surface of the left hemisphere, pretty well back toward the tentorium. The cortex was ruptured in this location, and a gelatinous, bloody mass escaped. The rest of the encephalon seemed normal to external inspection.

A vertical transverse section was made through both hemispheres in the motor area, passing through the ascending frontal gyri. Occupying the centrum ovale underneath the left cortical motor area, and completely undermining it was a large cavity capable of holding 100 cc. (?), very much resembling a distended lateral ventricle, which contained a large amount of coffee-red serum, and also a mass (tumor) lying on its inner side, near the paracentral lobule. The tumor was gelatinous and grayish-red. The walls of the sac were vascular and grayish, and appeared covered by an ependyma-like membrane, which, under the microscope, was found to consist of capillaries and portions of blood pigment.

The tumor itself had formed a connection with the falx cerebri posteriorly, in the region of the paracentral lobule,

and this region of the cortex was thinned; it bulged across the median line and indented the opposite hemisphere.

On the left side the corpus callosum was pressed downward, and the optic thalamus was also depressed and flattened. The left lateral ventricle was displaced downward and closed by pressure; on opening it, it was found free from disease. These appearances were sketched from the fresh surface of section by Dr. Amidon, and are shown in fig. 3.

Sections made through the hardened brain confirmed the above notions of the seat of the tumor. It lay wholly be-

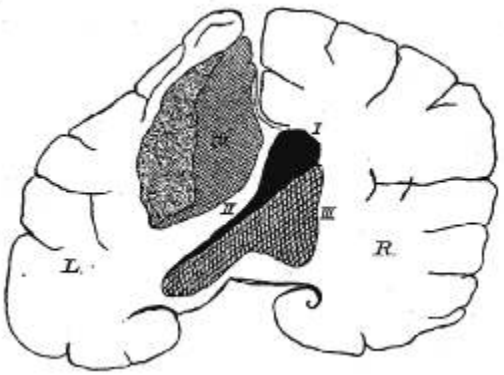

FIG. 3.

Transverse vertical section of the brain, Case 2, viewed from behind. *R*, normal right hemisphere; *L*, diseased left hemisphere; *I*, distorted lateral ventricles; *II*, depressed corpus callosum; *III*, thalami optici, depressed on left side; *IV*, the tumor; *V*, the cavity formed by the hemorrhage.

neath the externally visible convolutions of the left hemisphere, springing from and destroying that part of the first frontal gyrus which lies within the longitudinal fissure, above the corpus callosum and the paracentral lobule, forcing downward the gyrus fornicatus, extending outward into the white substance of the hemisphere, causing great compression of the surrounding parts, including the upper extremities of the first and second frontal gyri, the upper half of the ascending frontal and parietal gyri, and, to a less extent, of the upper parietal lobule.

A part of this pressure was due to the cyst lying outside of the tumor, near the convexity convolutions, which is more especially shown in the sketch made by Dr. Amidon from the fresh specimens.

The situation and dimensions of the lesion in this second case were therefore very different from those in the first case. In Case 2 the destructive effects of the tumor were expended upon the gray and white substances lying next

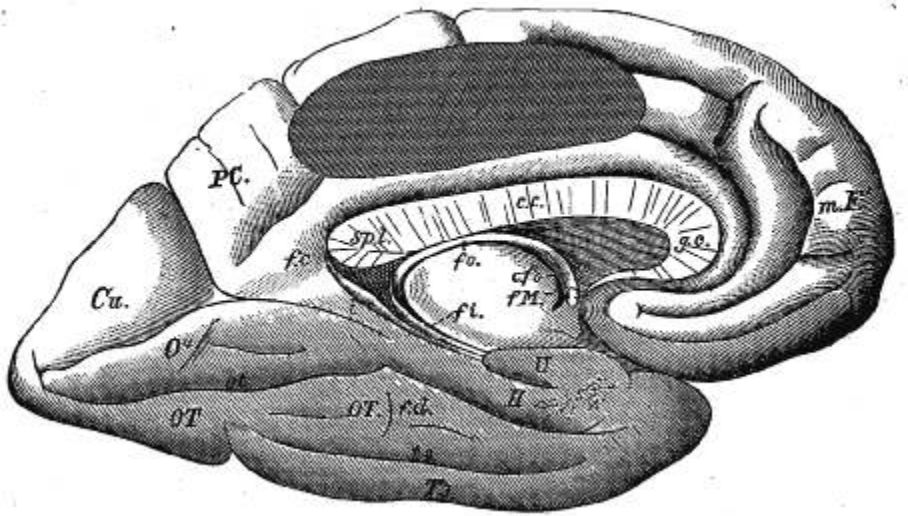

FIG. 4.

View of inner surface of the left hemisphere, after Schwalbe. Shaded spot indicates the superficial location of the tumor.

the longitudinal fissure, and the rest of the hemisphere suffered only compression effects. The posterior extremity of the intra-fissural part of the first frontal convolution and the paracentral lobule suffered the most destructive effects.

The tumor and cyst were of very irregular shape and I can only give approximate measurements. In the longitudinal fissure and near it in the brain the tumor was about 60 mm. in length (antero-posterior dimension); on a verti-

cal transverse section of the hemisphere, as in fig. 5, it measured 30 mm. transversely, and from 30 to 35 mm. vertically. These figures include the cyst, which was more developed in the frontal lobe, extending forward as far as the posterior part of the second frontal gyrus (wholly under it). The other (posterior) extremity of the lesion, the

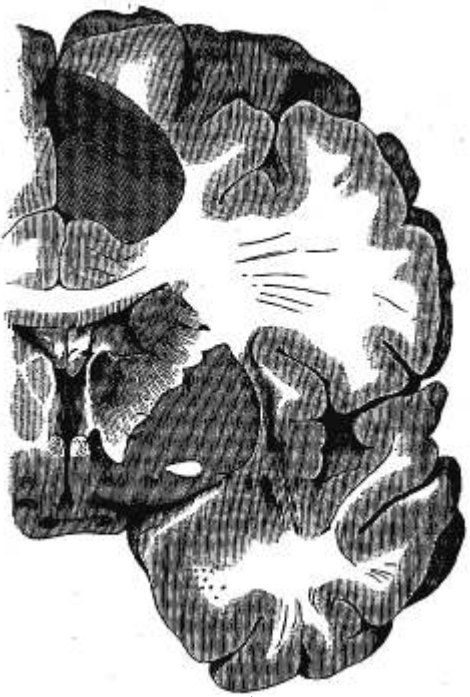

FIG. 5.

Transverse vertical section of left hemisphere, anterior view; after photo. No. 4 of Bitot. Shaded spot in upper part of drawing shows the location of the solid tumor.

solid growth, could be traced, on the median surface of the hemisphere, well into the surface of the precuneus.

A microscopical examination of the tumor showed it to be a common small-celled sarcoma.

Remarks.—There are many interesting features in the

semeiology of these two cases, but I shall dwell only upon those symptoms which are concerned in the questions of cerebral localization.

In both cases the first motor symptoms were epileptiform, and in Case 2 the spasm was the first and only symptom for many months. In Case 1 it was preceded by severe pain in the occipital region. In Case 1 attacks of jerking of the left arm, as well as trembling of that member, were observed by the patient some weeks before the weakness became apparent. There was no jerking of the cheek or leg. It was a brachial monospasm. It is remarkable and most instructive to note how quickly paresis and paralysis followed, these phenomena being for a long time limited to the arm; a brachial monoplegia succeeding the brachial monospasm. Contracture of the arm and hand also showed itself, but at what time is not definitely stated.

Late in the disease, when she came under observation, the left lower face and left third nerve were somewhat paretic, the sensibility was somewhat impaired on the left side of the body.

If it be permissible to formulate the chief symptoms observed during life in correlation with the lesion found *post mortem*, then this (Case 1) was a remarkable instance of irritating and destructive lesion of the upper part of the right ascending frontal gyrus, causing brachial monospasm and brachial monoplegia on the left side (with other phenomena of secondary logical value).

In Case 2 the course of the motor phenomena was quite different. There was a period of two years previous to the patient being seen by me, in which the only symptom was right hemi-epilepsy. That is to say, from time to time clonic epileptoid spasms occurred in the right arm and leg for a few moments. The face was never affected, the patient could usually talk in the paroxysm, and he only once

lost his consciousness. He was unable to say whether the spasm appeared first in the arm or in the leg.

At the time when the patient presented himself at the hospital the paralytic phenomena were just developing. He was still working all day at his trade, and was not conscious of the partial hemiplegia. This was, and remained until the apoplectic attack, more marked in the lower than in the upper extremity. At the time of first examination the right hand (affected side) was still stronger than the left hand, but the walk was slightly hemiplegic, the right foot being held in a slight equino-varus position. There was then no facial paresis and no aphasia.

Later the epileptiform attacks were controlled by bromide of potassium, but the hemiplegia progressed, still greater paresis being noted in the lower extremity.

About six weeks after first calling at the hospital, the patient was stricken down by an apoplectic attack, which rendered the right hemiplegia complete in the arm and leg, with marked paresis of cheek, but never aphasia. This attack obscured the symptoms which we may reasonably assume had been caused by the tumor. At no time was there marked anæsthesia on the paralyzed side.

Headache was remarkably slight prior to the occurrence of the apoplectic attack.

The *post-mortem* findings explain all these symptoms very well I think. The cyst outside of the tumor proper, found in the white substance of the hemisphere, was the remains of a hemorrhage which took place at the moment of the apoplectic attack, which was characterized by intense pain in the head, vomiting, collapse, and complete right hemiplegia. Dr. Amidon states that in removing the brain a small laceration occurred, and "a gelatinous bloody mass escaped," probably the contents of the cyst, about seven weeks old.

The long stage of hemi-epilepsy without paresis, two years, is accounted for by the fact that the morbid growth began upon the median surface of the hemisphere, springing from the pia covering the inner winding of the first frontal gyrus, and perhaps the paracentral lobule; at any rate, for a long time it was an *irritating lesion* causing discharges, and only gradually exercised enough pressure to *destroy* the irritability of the neighboring gyri. The parts of the hemisphere which must have suffered first in a *destructive* manner were the paracentral lobule and adjacent parts (posterior extremity of first frontal gyrus on median surface), and in connection with this should be noted the fact that paresis of the leg preceded and preponderated over that of the arm, until the apoplectic attack occurred. The absence of aphasia throughout, and of facial paresis previous to the hemorrhage, are likewise of interest.

If I may venture to formulate this case, I should define it as one of *irritating and destructive* lesion of the left paracentral lobule (and adjacent parts), causing crural and brachial monospasm and monoplegia, with greater development of symptoms in the leg.

It will be seen by a reference to the now numerous recorded cases of localized cerebral lesions that the two cases which I report are in sufficient harmony with the results reached, by many observers, thus far, viz., that the "centres" for the hand and arm are in or about the ascending frontal gyrus in its middle region, while the "centres" for movements of the lower extremity are further backward in the posterior extremity of the ascending frontal and ascending parietal gyrus, and their prolongation upon the median surface of the hemisphere, known as the paracentral lobule.

The many other interesting features of these two cases of cerebral tumor, I purpose considering in a future article upon the semeiology of cerebral tumors in general.

Before closing this contribution, it may perhaps be well if I present a brief *résumé* of the other cases of localized cerebral lesion which I have thoroughly studied (*i. e.*, while alive and *post mortem*) since the publication of my first paper on localization. Most of these cases have been published in medical journals.

CASE 3.—Hemiplegia with first symptoms in foot, and a limited cortical lesion.

In November, 1878, I saw, in consultation with Dr. Granniss of Saybrook, Ct., a gentleman aged 54 years, who was hemiplegic on the left side, and almost unconscious. The following account of his illness was furnished:

In December, 1877, after having enjoyed good health, he awoke one night with clonic convulsions of the left toes, foot and leg only. There was no impairment of consciousness, no spasm in any other part. He watched the spasm some time, and made comments on it. Since, there has gradually developed a left-sided hemiplegia. For months only the foot and leg were paretic; in the last few weeks the left arm has become weak, and now the left cheek is paretic, though the relatives have not noticed it. In January, 1878, vision became impaired, but an examination by Dr. Noyes revealed no cause. In the last few weeks patient has seen double at times, and sight has gradually failed. Severe headache has existed from the first; frontal, bilateral pain, most marked on the right side. The pain has been worst about daylight. In the past month pain decidedly nocturnal. On a number of occasions "lost himself" while out of doors, not remembering where he had been (petit-mal?). A business associate thinks that patient has committed errors in judgment. No extravagance in design or in deed. Lately has become stupid and semi-comatose.

Since January, 1878, a tumor-like swelling has appeared over the right parietal region. No albuminuria, but has had several attacks of gout. After severe cross-examination, patient admits having had a chancre fifteen years ago, treated with mercury; denies secondary and tertiary symptoms.

Examination showed a typical left hemiplegia, face and limbs. No diplopia, pupils small and equal; after atropia there is found a well-marked double neuro-retinitis. Sensibility preserved on the paralyzed side. Articulation indistinct, no aphasia. Stupor is peculiar, like that of drunken sleep. Patient can be roused by

loud talking and shaking, and then answers correctly (showing fair memory) and clearly. The swelling upon the head, raised perhaps half an inch, is just above the right parietal eminence, extending inward to the median line, and forward almost to the vertical line from the meatus auditorius to the bregma. This tumor overlies Ferrier's centres for the leg.

Diagnosis: External and internal nodes involving dura mater and the subjacent gyri of the right hemisphere.

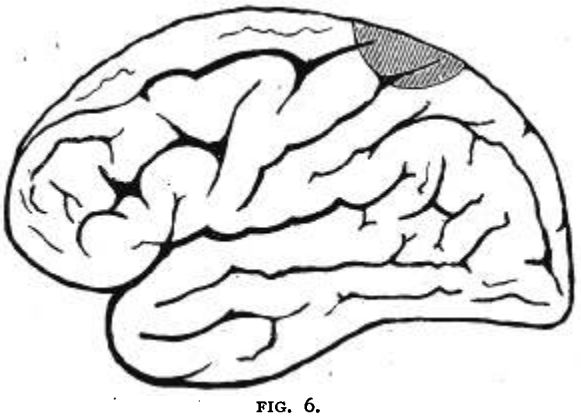

FIG. 6.

Lateral view of right cerebral hemisphere, with lesion.

A few days later the patient died comatose, and after much trouble Dr. Granniss secured a partial autopsy. He was not allowed to raise the brain from the skull or to incise it. He simply removed the calvarium and noted the lesions at the vertex. He found that there was an internal as well as an external osteitis, forming quite a tumor which had, after adhering to the dura, exerted great pressure upon the subjacent convolutions of the right hemisphere. Dr. Granniss marked the location of the cortical lesion upon an Ecker's diagram, and the annexed wood-cut is a copy of his sketch.

It is of course very much to be regretted that a thorough examination of the brain was not permitted, but in view of numerous recent cases, it is impossible not to admit a causal relation between the lesion causing pressure upon the inner end of the right ascending frontal and parietal convolutions and the symptoms in the left foot and leg—spasm and paralysis.[1]

[1] *Archives of Medicine*, vol. ii, p. 105. (A remarkable case of hemorrhage under the paracentral lobe, with paralysis of the opposite leg, is recorded by Dr. Miles, of Baltimore, in the same journal, p. 103.)

CASE 4.—Aphasia with word-deafness; no permanent paralysis; lesion in the parietal region.[1]

The main facts of the last illness of the late Dr. C. M. A., of New York, are already well known to his numerous friends in the medical profession, who watched the progress of his disease with painful interest. Throughout his illness he was attended by his partner, Dr. A. Dubois, and myself. He was also seen in consultation by Profs. Austin Flint, Sr., John T. Metcalfe, H. D. Noyes, and Dr. Allan McLane Hamilton; and for several months was under the professional care of Prof. E. C. Seguin.

Dr. A. was born in 1827, and was therefore fifty-two years of age at the time of his aphasic attack. At the age of eleven years he had a long illness, which was called "brain fever." Whatever may have been its real nature the illness was sufficiently severe to seriously endanger life, and for several years retarded his growth. At about the age of thirty years he had an attack of inflammatory rheumatism affecting the larger joints. This was followed by three or four other attacks within the next few years, but none of them lasted longer than from three days to a week, or was attended, so far as we can learn, by any cardiac complication. Twelve years ago he had a well-marked attack of gout, and since then from three or four other paroxysms, the most severe one five years ago, after a violent quinzy, when both great toes were affected. For several years before his aphasic attack, he was subject to flatulent dyspepsia, and had occasional outbreaks of eczema. It should be noted here that neither gout nor rheumatism were hereditary in his family, and that the most frequent cause of gout —over-indulgence at the table—was notably absent in his case, as he was usually very abstemious both in eating and drinking. In November, 1877, he had a severe attack of renal colic. The concretion was arrested in the ureter, and not discharged until the end of ten days, after repeated paroxysms of colic. The stone, on analysis, was found to be composed of uric acid. On February 1, 1879, he attended a concert in evening dress, and on his way home became thoroughly chilled. During the night he was awakened by pain and oppression in the chest, these symptoms continuing during the following day. As there was no evidence of pulmonary lesion, but merely tenderness over the middle portions of the chest anteriorly, on both sides, with pain in these situations on movement of the pectoral muscles, the symptoms were

[1] Dr. A. B. Ball. A contribution to the study of aphasia, etc. *Archives of Medicine*, vol. v. No. 2, April, 1881.

referred to muscular rheumatism. Within a few days he was able to return to business, but was still so far from well that some more serious disturbance was apprehended by his medical attendants.

On February 11th, the date of his aphasic attack, he was in much better spirits. At half-past eight in the evening he was seen in his office writing a letter. A few minutes before ten o'clock he rang his bell violently, and was found by his servant lying on the lounge talking unintelligibly. I saw him not more than five minutes afterward. He was conscious, but unable to answer questions except by a confused muttering. The face was slightly flushed; pulse soft, easily compressible, about 90 per minute; the first heart sound feeble, and no murmur audible. Incomplete right hemiplegia and right hemi-anæsthesia. Was apparently aware of the nature of his attack as he pointed to his right arm and left frontal region. By gestures he finally succeeded in directing my attention to important cases in his note-book, requiring attention on the following day. At eleven o'clock he was seen by his partner, Dr. Dubois, and with slight assistance walked up two flights of stairs to his bedroom. On the following morning he complained of paroxysms of pain in the left frontal region. This symptom, which yielded to local applications of hot water, annoyed him frequently for several weeks, and recurred at intervals during the whole course of his illness. Repeated examinations of the heart failed to disclose any morbid condition except feeble action and moderate hypertrophy. No albumen or casts in the urine. Absence of fever, except on the evening of the third day, when there was a slight rise of temperature which lasted only a few hours. From this time his physical condition steadily improved, and by the end of six weeks his general health was fairly restored. Beyond slight paresis of motility and sensation on the right side the only marked change was the aphasic condition to be presently described. During the summer and autumn of 1879 his physical condition remained fairly good. The kidneys performed their work well, although it was evident from the occasional appearance of traces of albumen and casts in the urine, and from the enlargement of the left ventricle without valvular murmurs, that the kidneys had probably undergone cirrhotic changes. At no time was any increased arterial tension noticed in the sphygmographic tracings, but this absence was ascribed to muscular degeneration of the cardiac muscle, as feeble action of the heart was a constant symptom throughout his illness.

In March, 1880, he had another attack which was supposed to

be due to a small cerebral hemorrhage. At dinner, while talking with a friend, he suddenly turned his head to the right, and began muttering incoherently. With assistance, he immediately left the apartment and walked to his bedroom, muttering all the way with his head turned to the right. At my visit, half an hour later, when his consciousness was fully restored, he said that the attack began with an explosive noise in the head like a pistol-shot. Immediately he heard some one talking to him over his right shoulder, and turned to see who was addressing him.

Every word uttered by himself, he said, was mockingly repeated by this imaginary individual, and the mutterings his friends had heard were his indignant protests against the insult. On examination there was found slight paralysis, with numbness and anæsthesia on the *left* side. These symptoms disappeared after a few days, his mental condition remaining without apparent change. Shortly after this attack it was evident that his heart was failing in power. He frequently complained of breathlessness on exertion, and the heart sounds were feeble, with occasional intermittence of beat. Toward the end of May he was seized with what proved to be his final attack. The symptoms were slight fever for several days; oppression in the chest with shortness of breath; slight cough, generally dry but occasionally accompanied by expectoration tinged with blood; and marked tenderness over the region of the heart. At a few examinations a faint aortic obstructive murmur was heard, or rather a soft blowing sound over the base of the heart near the aortic valves, with the first sound. Urine nearly normal in amount; specific gravity varying from 1012 to 1018; no albumen and no casts except a few hyaline cylinders found at one examination. These symptoms were hardly sufficient to warrant a positive diagnosis, but they seemed to point to endocarditis with possibly myocarditis, and this view was confirmed, or at least considered plausible, by Prof. J. T. Metcalfe, who saw him in consultation. The urgent symptoms subsided by the end of a week, but he was still much prostrated, and complained of giddiness and mental confusion. On one occasion he exhibited in a marked form the so-called rotatory phenomenon, turning over rapidly to the right, and would have rolled out of bed had he not been prevented. On June 19th, about 3 P.M., he suddenly became totally blind. Dr. Dubois, who saw him shortly afterward, found him still partially blind, but gradually regaining his vision. At my visit, two hours later, he was perfectly conscious, with his sight fully restored. Half an hour afterward he fell into a quiet slumber, from which he

suddenly awakened at 7 o'clock, exclaimed "Oh!" and died instantly.

In considering the aphasic symptoms which constituted the most striking and interesting feature of his case, a few preliminary remarks on the essential nature of aphasia may be permitted before analyzing the symptoms in detail.

The interchange of thought between members of the human family is carried on by means of various symbols, that is, by signs which stand for the ideas they represent; for example: articulate sounds, written language, gestures, facial expression, mathematical, musical, and other signs. In aphasia this symbolic function, or capacity to *interpret* and *express* thought in a symbolic form—the *facultas signatrix* of Kant—is more or less seriously impaired. In some cases the chief difficulty is in the direction of *symbol-expression* (ataxic aphasia), the concept being present, but failing to enunciate itself on account of some lesion in the motor track concerned in the expression of symbols. In other instances the concept is present in the mind, but the appropriate symbol for it is forgotten (amnesic aphasia). In a third class of cases there is also a defect in the capacity for *comprehending* symbols. Certain auditory and visual impressions, especially those of word symbols, fail to recall into consciousness their corresponding concepts, although the capacity for forming such concepts under the influence of other stimuli may still be retained. When concepts can no longer be formed, the lesion involves the fundamental processes of thought, and extends beyond the sphere of simple aphasia. The latter term fails, however, to recognize the impaired capacity to *understand* symbols, and as most cases of aphasia present some degree of this derangement, Finkelburg[1] has proposed to substitute the word "asymbolia" as a generic term for all the phenomena of

[1] *Berl. Klin. Wochenschrift*, 1870, Nos. 37, 38.

aphasia. Kussmaul[1] prefers the term *asemia*, suggested by Steinthal, as being still more comprehensive; "symbol" represents an idea behind it, whereas "sign" often represents merely an emotion. In the following description of the aphasic symptoms in Dr. A.'s case, we shall use the word "symbol" in preference to "sign," as there was no difficulty in comprehending or expressing emotions. Our classification is based upon that of Spamer.[2]

I.—EXPRESSION OF SYMBOLS.

a. Disturbances of speech. On the morning following the first paralytic seizure, by which time the general shock to the brain had abated, it was evident that the cerebral disturbance was limited chiefly to the *verbal* expression of ideas. His general intelligence was fairly well preserved, and he understood much that was said to him, but there was a marked defect in verbal expression. His principal difficulty was with proper names and common nouns. When a glass of milk was held before him, he said: "That is something to drink," recognizing at once its several attributes, its color, uses, etc., but the word which combined these qualities into a single concrete expression, or symbol, he could not utter, even when the word was repeated to him. He had less difficulty with adjectives, verbs, and adverbs, that is, with words of less concrete symbolic character. His vocabulary of proper and common nouns very soon began to increase. Within the first few days we succeeded in teaching him a number of such words by directing his attention to the movements of the lips and tongue in pronunciation. My own name, being short and easily pronounced, he learned in one day, and rarely afterward forgot it. Long names of individuals, or long words which he

[1] *Ziemssen's Cyclopædia of Medicine.* American edition, vol. xiv, p. 609.
[2] C. Spamer. *Archiv für Psychiatrie*, Bd. vi, p. 526.

rarely had occasion to use, he seldom mastered completely at any period of his illness. During the summer and autumn of 1879, his vocabulary increased so as to include a considerable number of words used in ordinary conversation. With these he generally succeeded in expressing his ideas fairly well, but an attempt to leave the beaten track resulted in mental confusion and inability to proceed with the conversation. In rare instances his conversational powers astonished his friends, and gave him delusive hopes of ultimate recovery. On one occasion he conversed with fluency on various topics for nearly an hour, with a friend who had not met him for several years and was unaware of his illness. His friend noticed no aphasic disturbance during the interview, and was greatly surprised afterward on learning the facts of the case. Such flashes were, however, only intermittent, and it became more and more evident that any thing like perfect recovery was hopeless.

In conversation, true *paraphasia*, that is, the substitution of wrong words, was rarely noticed. Almost invariably the word uttered bore some resemblance to the correct one, and differed from it in only some of its letters. Thus the first letters were usually correct. This fact was of great assistance to him in conversation, as it enabled him, when he knew the first letter, to find the correct word in a dictionary or work of reference, *where he at once recognized it as soon as he saw it*, showing that the concept was present in his mind in a latent form, and needed only the right stimulus to recall it into consciousness. His Medical Register was frequently consulted for physicians' names he was unable to pronounce, as he retained, to a marked degree, his interest in news affecting the medical profession.

In the expression of musical and other non-verbal sounds, as in singing, whistling, and imitation of various significant sounds, there was no observable deficiency.

As regards the *alphabet* and *numerals* the same cannot be said. At the outset of his illness he was able to pronounce only a few letters, and could not count above four. With training, however, he in time learned most of the alphabet, but never succeeded in spelling any but short and simple words. Counting he reacquired quite perfectly, and was able to solve simple sums in arithmetic, that is, to express their answers verbally. Even when unable to do this he could often *write* the answers correctly. When both these efforts failed him he was frequently able to recognize the correct answers if shown to him in writing. During the latter part of his illness he supervised his business accounts, and rarely failed to notice mistakes in them made by others. This circumstance belongs, however, rather under the head of symbol-*comprehension* than under that of symbol-*expression*.

b. Defects in writing. At the outset of his illness there was complete *agraphia*. When asked to write the word "cat," he took the pencil in his left hand, and drew three perpendicular lines, naming them one, two, three. As we shall see in a later illustration, this substitution of numerals for letters and words was at first very noticeable. He knew the number of letters required for the word "cat," but there was *no attempt at the formation of letter symbols*, although he was perfectly aware that his straight lines were not letters. Under training he gradually learned to form letters with his right hand, and after several months could copy simple sentences correctly, sign his name in his usual clear and elegant handwriting, and even write short sentences of his own composition, but more than this he never succeeded in accomplishing.

c. Gesture language. The capacity for expressing ideas by gestures seemed to be unimpaired. He retained much of his natural vivacity of manner, more in fact than could

have been expected in a person of his keen sensibility, when he found himself cut off from the ordinary modes of social intercourse. His gesture language had always been a prominent characteristic, and now became an important aid in the expression of ideas. Names of individuals and objects, which he was unable to remember or to pronounce, he frequently succeeded in recalling to others by gestural description, and this was very noticeable even early in his aphasic attack.

II.—COMPREHENSION OF SYMBOLS.

Before entering upon this branch of our subject it should be noted that the senses of sight and hearing in the present case were perfect, so far as could be determined by the usual tests. With respect to vision, the only exceptions to this statement were a transient attack of total blindness a few hours before death, and occasional attacks of hemiopia. Prof. H. D. Noyes, who made an ophthalmoscopic examination of his eyes in the autumn of 1879, reports that "he found no remarkable change in the optic nerves or retinæ. The arteries of the nerves were rather small, and, with this exception, nothing abnormal was noted."

A.—*Comprehension of Auditory Symbols.*

a. Spoken words. Early in his illness, on my remarking to him one day, "Dr. Peters called to see you," he replied, "I don't know him." The name was repeated several times, but he failed to recognize it, although it was the name of an intimate friend. The written name was then shown him. "What a fool I am," he exclaimed, "of course I know him." This was the first instance in which my attention was drawn to the fact that certain auditory impressions failed to be converted into concepts, although the conceptive faculty remained intact. Not long afterward he noticed

this peculiarity himself, as was shown by his remarking to me: "The words I can't pronounce are the words I can't *hear*." This observation, the general correctness of which was verified by repeated experiments, points to a very interesting peculiarity in his case. The words over which he stumbled in conversation were words which made no intelligible impression on his mind when repeated to him, and, conversely, the words he failed to understand in conversation were words he had great difficulty in pronouncing spontaneously. The concepts represented by these word symbols we were generally able to recall to his consciousness by other means, such as writing, gestures, etc., but even then he was unable to express them, except after a certain amount of training. This "word-deafness," except when it was possible to stimulate the conceptual centres by visual or other impressions, made it extremely difficult to determine how much of his aphasia was due to the *ataxic* and how much to the *amnesic* element.

b. Musical and other sounds. His appreciation of music was fortunately well preserved, and was a source of much pleasure to him. In attending concerts and operas he exhibited his usual good critical taste. The significance of other sounds, such as the tone of a bell, the striking of a clock, etc., was perfectly understood.

B.—*Comprehension of Visual Symbols.*

On the third day of his aphasic attack a scroll of Scripture texts was held before him, and he was asked to read the following sentence: "We love Him because He first loved us. While we were yet sinners Christ died for us." He read aloud as follows: "We he have two three that I have to have the same. I have two three." The substitution of numerals for words is here again noticed as in a previous illustration. The words "the same" probably refer to the

repetition of "love" in the first sentence. He was aware that this rendering of the text was incorrect; in fact he almost always knew when he read aloud incorrectly, and expressed impatience thereat. Later in his illness when he was able to read sufficiently well to gather from the newspaper the main points of news, he remarked to me that there were always words in every long sentence which conveyed no impression to his mind, and that he was compelled to form his idea of the meaning of such a sentence from the other words whose meaning he understood. The significance of many of these uncomprehended words could be conveyed to him in other ways, showing that his failure to recognize the written symbols was not always due to a defect in the conceptual centre, but rather to a lesion in the channel of transmission from the optical centre for word symbols to their ideational centres.

The same difficulty extended at first also to the comprehension of *written numerals and their combinations*, but, as we have already seen, he reacquired, to a certain extent, this capacity under training. *Gesture language* he understood perfectly from the start.

The degree of impairment in intelligence, otherwise than in the comprehension and expression of symbols, it was extremely difficult to determine, for reasons already given. His intimate friends were satisfied that there was much less general mental deterioration than those who met him casually would infer. His memory of incidents in his own life, of the past illness of his patients, and of numerous other details was strictly accurate, so that we could rely upon his statements upon such points in every particular. In business matters he always manifested his usual tact and good judgment. During the last few months of his life he was a constant attendant at the surgical operations of the New York Hospital, of which he was an attending surgeon, and

his criticisms showed that he retained not merely a general interest, but also his special knowledge in surgery. On several occasions he assisted me in minor surgical operations and dressings, with his usual deftness and attention to details. At whist, euchre, and all games with which he had been familiar, he was as expert as ever. During the winter of 1879–80 he consulted numerous medical works on the subject of aphasia. Since his death I have seen a sheet of paper containing his notes of reference to articles on this subject in English and French works and journals. The titles, dates, etc., are strictly correct, and are written in his usual clear and elegant handwriting. His memory of location was particularly well preserved. He could always turn without hesitation to the right place in books he wished to consult, remembered the houses of friends—that is, their relative positions in this city,—and in numerous other ways showed that he perfectly understood the spatial relations of objects. The only exception to this fact was a singular symptom which annoyed him for several months, viz.: a tendency to reverse the natural position of objects which he handled, such as table-knives, spoons, pencils, canes, etc. He immediately recognized his mistake, however, and corrected it, but always spoke of the inclination as irresistible.

As an aid to the interpretation of the aphasic symptoms in the present case, we reproduce, below, Spamer's diagram representing the several tracts between the reception of impressions, the comprehension of these impressions, and their expression.

It will be noticed in the above diagram that the tract from P to B is represented by a straight line, while the tract from P' to B pursues a circuitous route. By this distinction Spamer attempts a rough explanation of the difference observed in most cases of aphasia between the comprehension of *auditory* word symbols and the comprehension

of *visual* word symbols. Cases of marked word-deafness without ordinary deafness, seem to be extremely rare; at least there are very few instances of this kind on record. The tracts for *all* auditory impressions, he supposes, lie in close connection, and may be represented by a single

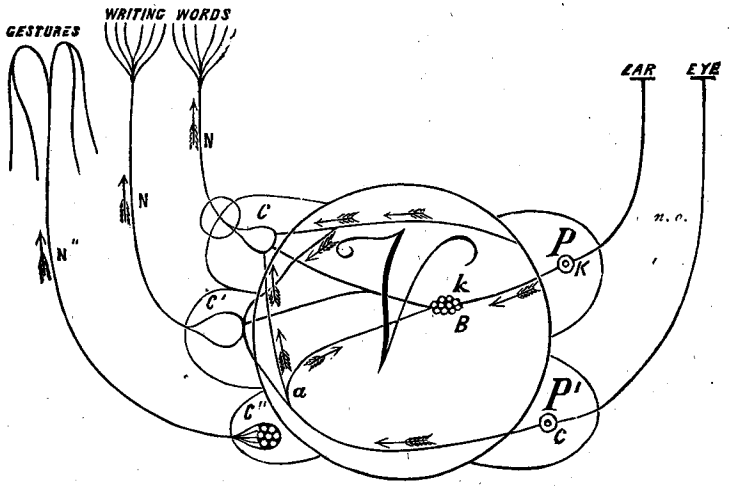

FIG. 7.

The circle in the middle of the diagram, *V*, represents the ideational tracts. From the right the excitations of the sensory nerves pass into the brain.

n. a.=auditory nerve. *n. o.*=optic nerve.

P and *P'* represent the places where the auditory (*K*)and the optical (*G*) impressions are perceived. When the impressions reach these points we have merely sense-perceptions without associated conceptions. The association with definite corresponding conceptions takes place only when the excitation travels onward to *B*, the conception. From this point the excitation may proceed to *C*, *C'* and *C"*, the centres of coördination for movements in speech, writing and gestures.

N, *N'* and *N"* are the motor nerves concerned in symbol expression (speech, writing, gestures). At their termination these nerves are broken up into fibres distributed to individual muscles.

The diagram represents the reception and tracts of *word symbols* through the eye and ear. The tracts of other auditory and ocular impressions are not designated.

straight line. With visual impressions the case is different. Aphasic patients very generally recognize material objects, but exhibit a marked defect in understanding written and printed words, as well as in expressing the concepts in speech and writing. The tract for visual word symbols is

therefore more or less widely separated from the tract for other visual impressions, and lies in some parts of its course near the centres of coördination for speech and writing, or near the tract from *B* to the latter. This explanation is ingenious, but hardly satisfactory. If the tract from *P'* to *B* should be represented by a circuitous route, that from *P* to *B* could scarcely have been *direct* in Dr. A.'s case, because the word-deafness was even more marked than the word-blindness, although both auditory and visual impressions, with the exception of word symbols, were interpreted with equal acuteness. Indeed, our main reliance, when the word symbol failed to be recognized by him in conversation, was to present the word to him in writing. The reverse process, that is, the presentation of the auditory, in place of the visual, word symbol rarely succeeded. In other words, he seldom understood the spoken words when he failed to comprehend the written form.

DR. SEGUIN'S REPORT OF THE AUTOPSY.

The autopsy was made twenty hours after death, on June 20th. The body was well preserved in ice.

Head.—The dura mater is abnormally adherent to the calvarium, on both sides equally; no thickening of dura. Pacchionian bodies small. Marked subarachnoid effusion, which has gravitated to posterior regions. Dura of base normal. The basilar artery is really a continuation of the right vertebral artery; the left being only 1 mm. thick. The right vertebral and the basilar arteries are the seat of patches of arteritis, separated by regions of healthy tissue, but nowhere obstructing the flow of blood. Circle of Willis is complete and patent. The carotids, just below the circle of Willis, are extraordinarily thickened, quite rigid, but not calcareous; their wall is nearly 1 mm. thick. The same alterations in patches can be traced in the accessible branches

of the middle cerebral arteries; the anterior cerebrals are only slightly affected. Nerves at the base normal. The left hemisphere is the seat of a large depression caused by the destruction of several convolutions, viz.: the whole of the inferior parietal lobule, with the first tier of temporal gyri. The posterior extremity of the angular gyrus, and the whole of the ascending parietal, are preserved. This lesion is a yellow patch lying in the region supplied by the terminal branches of the left middle cerebral artery.

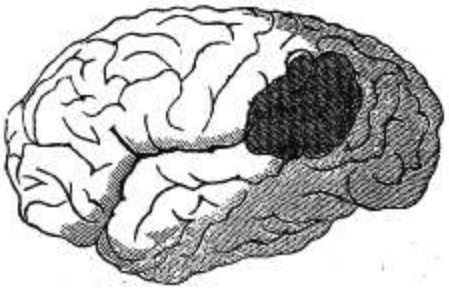

FIG. 8.

Lateral view of left cerebral hemisphere, after Henle. Shaded spot shows the superficial location of the yellow patch.

To external examination, the remaining convolutions are normal, more especially the third frontal, the ascending frontal, and the anterior gyri of the island of Reil. The first branch of the middle cerebral artery on the left side is pervious, though there are a few patches of arteritis near its origin. The main trunk of the artery, in the fissure of Sylvius, and its two terminal branches are pervious to the confines of the patch, and in the pia covering the patch. The patch was probably caused by blockade of smaller arteries which cannot be traced. The right hemisphere presents a healthy surface. On opening the fissure of Sylvius, the middle cerebral artery is found patent but bearing a few patches of thickening.

The brain is sliced in transverse vertical sections.

Section No. 1, about 37 mm. (one and a half inch) from apex of frontal lobes, presents no lesion.

Section No. 2, at a distance of 25 mm. behind No. 1, passing through the posterior extremity of the third frontal convolution and cutting off the apex of the temporal lobe, is free from lesion.

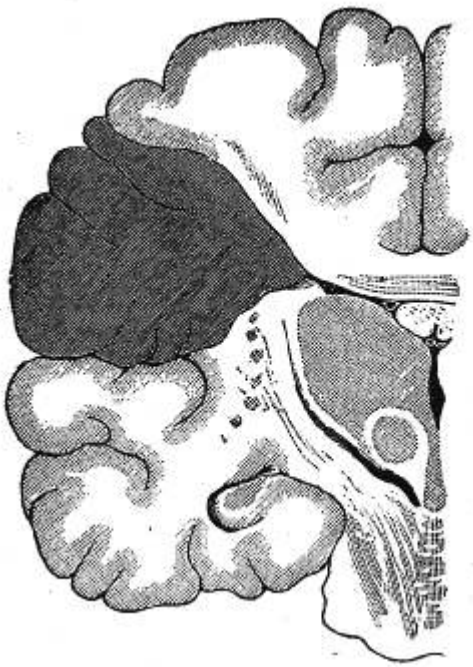

FIG. 9.

Diagram of transverse vertical section through left hemisphere, showing the extension inward of the patch. This view corresponds to section No. 4, described in the text.

Numerous fine slices made in the speech tract in this region (left side) reveal no alterations of structure.

Section No. 3, 25 mm. further back, showing the lenticular ganglion and the thalamus, no lesion.

Section No. 4, made at 25 mm. behind No. 3, passing

through the anterior limit of the yellow patch above described, and cutting through the posterior extremity of the thalami. There is no lesion to be seen except the yellow patch in the left hemisphere, and its full extent is well shown; besides destroying the convolutions it extends deep into the white substance of the hemisphere to the roof of the lateral ventricle.

Section No. 5, made at a distance of 25 mm. posterior to No. 4, reveals the penetration of the yellow patch as just described.

Section No. 6 shows no lesion.

The brain was afterward finely sliced up without any other lesion being discovered.

Sections made at different points in the pons Varolii and medulla oblongata seem normal.

Cerebellum normal.

CONCLUDING OBSERVATIONS BY DR. SEGUIN.

Dr. A's paræsthesiæ and perversions of muscular sense were very curious. He referred his sensations of numbness on the right side to homologous regions in the hand and foot, viz.: the distribution of the ulnar nerve and that of the musculo-cutaneous in the leg and foot. In the right side, generally, the paræsthesiæ were of drawing up, or tightening, and as if a strong rotatory movement were going on in each limb around its longitudinal axis, the hand in pronation, the foot in inversion. The patient's account of these subjective movements never varied, and he would often illustrate them by moving his hand and forearm in extreme pronation and rotation.

The impairment of muscular sense of which he complained was something which I had never met with before. If he did not use his eyes in prehending objects with his right hand, he would find that he had seized them by the

wrong end. He sometimes found himself standing with the head of his cane on the ground and its point in his hand. Frequently, in my presence, he essayed to grasp a pen or pencil with his head turned away, and repeatedly he found himself holding the object by the wrong end, and this after turning it over three or four times to get its outlines.

Yet with these perversions of sensibility there was no common anæsthesia, either to pricking, to cold, or to æsthesiometer points.

Dr. A's aphasia was complex, but the striking feature in it, during my six months' observation, was the word-deafness.

He could express himself fairly well in short sentences, and might for a little while carry on a commonplace conversation with a non-expert without betraying his defect; but he frequently failed to find the right word, and often found it only after struggling a good deal.

In attempting to speak he would often, after failing to get the proper noun, use a corresponding verb or employ synonyms, showing that his idea or concept was always correct, but that his vocabulary was faulty. He could copy written or printed characters quite readily, but experienced great difficulty in writing spontaneously.

All the auditory relations of language were much impaired. He used to say that going to church and listening to a sermon was to him all a mixed-up, meaningless jargon, like "drub-arub-drub." He could catch very few words. In ordinary conversation, familiar short sentences were apprehended readily; equally simple sentences, containing other than the most commonplace words, had to be repeated again and again. Reading from a book was jargon to him. Writing from dictation was impossible, and even the alphabet was poorly executed in this way. The sound of the letter *c* seemed the one for which he was most deaf.

Yet his hearing was not impaired (I never tested it carefully), and he understood and appreciated music. While a lecture or a sermon was unintelligible, he enjoyed a concert and claimed to appreciate it. He whistled and hummed airs correctly—much better than he spoke.

I often questioned about and tested him for hemiopia, with negative results. Occasionally he had attacks of moving fortification lines in the left fields of vision, but these were evidently phenomena of the migraine type.

The pathology of the case is obscure in many respects.

The arteritis (see Dr. Peabody's description) is not of the senile type, and the patient's statement, that he had never had syphilis, was positive, and, we believe, perfectly trustworthy. This would, therefore, be one of the best authenticated instances of non-specific endarteritis deformans, leading to obliteration of the calibre of small arteries, ischæmia of a cerebral territory, and softening.[1]

The location of the lesion is peculiar, and some years ago would have been considered as destructive of the modern theory of aphasia. In view of the experiments of Ferrier, Munk,[2] and others, however, it seems clear that the lesion occupied a portion of the brain which is concerned in the reception of sensory impressions from various sources, more especially the eye and ear.

So long as aphasia was looked upon as sometimes a form of motor disorder, a difficulty in the emission of language, and in other cases as dependent upon verbal amnesia, it was impossible to explain its production by a lesion of the parietal or sphenoidal lobes. In the last two or three years the elements of imperfect perception of the written signs and spoken sounds of language—word-blindness and word-

[1] It is very much to be regretted that the cerebral arteries and the brain itself were not examined microscopically; but the autopsy was allowed only on condition that the brain be not retained for examination.

[2] Consult: Ferrier, The Functions of the Brain, New York, 1876; Munk, Ueber die Functionen der Grosshirnrinde, Berlin, 1881.

deafness respectively—have received some recognition, and these phenomena are perfectly explicable by lesions placed in the sensory or perceptive regions of the cortex and internal capsule.

In such cases the aphasia is indirect, not due to any interference with the channel for the emission of sound-forming impulses, but to a break in the other part of the circuit, viz., the receptive organ.

Dr. A. B. Ball, of New York, is the author of the article from which the foregoing large extract is made, and my small share in it is the description of the lesion found in the brain, and some general remarks upon the pathology of aphasia. In this connection I quote from the article because the lesion seems to indicate the postero-inferior limit of the motor area of the hemisphere. Although a large part of the inferior parietal lobule, and the first tier of temporal gyri, together with the associated white matter, were necrosed, there was no permanent hemiplegia. At the beginning of the illness, for a while after the attack, "slight paresis of motility" was noted. Whatever value this case may have for the study of indirect aphasia, it certainly will rank high as a negative case in the question of cortical motor localizations.

CASE 5.—Abscess of the left frontal lobe of the cerebrum, without motor phenomena.[1]

On April 11, 1880, I was asked by Dr. J. Lewis Smith to see a case in consultation with himself and Dr. J. R. Leaming. The patient was a young married woman, aged about 28 years, who had formerly enjoyed good health and had borne several children. During the month of February one of these children had died after a severe illness, and she had undergone considerable fatigue. She seemed depressed, weak, and anæmic afterward.

About four weeks before the date of consultation she complained of pain over the left eye. This was soon accompanied

[1] *Archives of Medicine*, vol. v, No. 1, Feb., 1881, p. 107.

by swelling and exophthalmus, and on March 24th Dr. Knapp was called in and diagnosticated orbital (sub-periosteal) abscess. This was opened on March 26th by Dr. Knapp.

It was remarked that the pus was under great tension, and that it spurted out a considerable distance when released. Pain ceased at once, the exophthalmus disappeared, and the wound quickly healed. During the first few days of April all seemed going on well; the wound was healed; the patient was free from pain; she was taking tonics, and on the 3d made a call on a near neighbor.

During the night of April 3d and 4th, one week before my examination, she awoke with severe headache and vomiting; ever since she has lain abed, presenting the following symptoms: headache, chiefly mastoid and through the base of the skull; occasional vomiting; irregular respiration; irregular and very slow pulse, varying from 60 to 50 beats per minute; stupor and general feebleness. As negative points there were no symptoms about the eyes, objective or subjective, except a partial ptosis of the left upper lid (which had been incised); no fever, chills, convulsions, paralysis, aphasia; at no time had there been coma. The urine was free from albumen.

Examination.—Patient was soporose, but could be roused by loud speaking; she answered questions as if half asleep, but in such a way as to leave no doubt as to the preservation of language. She put up both hands to the mastoid regions when indicating the seat of pain. A minute inspection showed no paralysis except about the left eye, whose upper lid drooped and whose internal rectus was inert. The pupil on the left side was not fully dilated, but it was a little wider than the right. The optic nerves appeared somewhat congested, and were dim at their periphery, but there was no actual choking. Patient appeared to feel pinching well everywhere. The thermometer showed no fever. The pulse varied from 53 to 66 beats per minute, and it was a reluctant, delusively full pulse, with no real strength. The breathing was easy and regular, but friends of the patient described quite well a Cheyne-Stokes breathing which they had observed. There was neither redness nor tenderness about the site of the orbital abscess.

I diagnosticated an abscess of the brain probably in the left frontal lobe, and expressed the opinion that the patient was in imminent danger. She died the next day in a comatose state; no new symptoms having been observed.

It was then learned that for two years Mrs. F. had suffered from frequent attacks of headache, lasting several hours. The pain was frontal, and sometimes extended along the nose and into the left temple. There had never been symptoms of chronic nasal catarrh.

The autopsy was made by me on April 13th, about thirty hours *post mortem*, in the presence of Drs. Knapp, J. R. Leaming, J. Lewis Smith (the attending physician), and Richard Wiener. We found a large abscess, the size of an

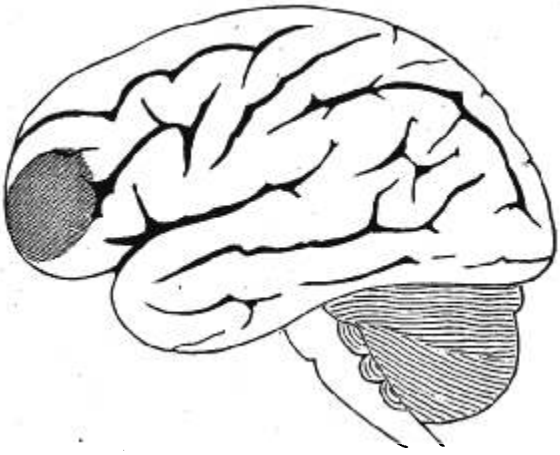

FIG. 10.

Apparent location of the abscess, drawn on an Ecker's diagram of the brain.

English walnut, in the left frontal lobe. It seemed to lie wholly under the cortex cerebri, in the convolutions of the orbital lobule, and in the second frontal convolution. Viewing the hemisphere from the side, the apparent posterior limit of the abscess was the anterior border of the lower part of the third frontal gyrus. Fig. 10 indicates the seat of the soft, fluctuating, bulging abscess. Its size and penetration were not then determined, as it was thought best to harden the brain as a whole, before making sections.

The external connections and origin of the abscess were most interesting. There was only one point of adherence between the diseased frontal lobe and the dura mater, and that was over the orbital plate of the frontal bone immediately under the swollen frontal lobe. There the dura mater was thickened and adherent to the pia mater and cortex cerebri, forming the inferior wall of the abscess, over a space as large as a ten-cent piece (about 15 mm.). Under this patch of pachymeningitis the orbital plate of the frontal bone was necrosed and perforated; a probe was easily passed into the orbit.

In the orbit, under its periosteum, pus was found, and a part of the roof and the inner wall of the orbit were carious. Careful dissection by Dr. H. Knapp showed disease of a similar kind in the ethmoidal cells and frontal sinus. I need say nothing more of the conditions of these parts and of the pathology of the orbital abscess, as the case has been fully reported from this point of view by Dr. Knapp.[1]

The appearance of the necrosed orbital plate and of the thickened, adherent dura mater, was precisely similar to what I have several times seen in cases of suppurative disease of the internal ear with cerebral abscess by contiguity. The genesis of the abscesses must have been alike in the two situations.

In December, the brain having been sufficiently hardened in bichromate of potash solution, I embedded it in Gudden's microtome, and made several horizontal sections through the whole brain with the view of demonstrating the relations of the abscess. These cuts showed that the abscess was of quite as large a size as at first supposed, almost perfectly globular in shape, measuring about 38 mm. in diameter. It contained ordinary pus, and was lined by a distinct membrane 1–2 mm. thick. The anterior,

[1] *Archives of Ophthalmology*, vol ix, p. 185.

inferior, and external limits of the abscess were thinned cortex and pia mater; superiorly, posteriorly, and internally, it was bounded by apparently normal white substance. The whole of the white centre of the frontal lobe, except a portion near the convexity of the hemisphere, was destroyed to within 10 mm. of the folds of the island of Reil, and about 8 mm. of the head of the nucleus caudatus. The mass of white substance connecting the inferior and posterior part of the third frontal convolution and

FIG. 11.

Relations of the abscess as shown in a horizontal section of the brain made at the level of Broca's speech-centre. Drawn from a photograph of the specimen. Occipital lobes cut off.

the anterior gyri of the island of Reil with the internal capsule, was uninjured.

This fact is of capital importance in estimating the bearing of this case upon the current notions of cerebral localization.

The above description of the topography of the lesion, especially its posterior limitation, is made from the surface exposed by the lowest cut made, viz., one passing through the speech-centre of Broca, about 10 mm. above the

apparent commencement of the fissure of Sylvius (pia still adherent). Fig. 11 is faithfully drawn from a photograph taken of this section-surface. The rest of the brain was healthy to the naked eye.

This remarkable case seems to me of much importance as a negative contribution to cerebral localization. It is in exact accord with recent experimental data, and with the *post-mortem* findings of the last ten years, that an abscess placed like this one should give rise to no motor symptoms, and should not cause aphasia. It is wholly within what are now called the inexcitable districts of the brain. The only symptoms present were the partial paralysis of the left third nerve (more immediately caused by the orbital abscess?) and signs of intracranial pressure. Yet it is important to note that in spite of the enormous pressure which must have existed there was no actual neuro-retinitis.

I have elsewhere reported another case of (smaller) abscess in precisely the same location (left frontal lobe) in which no symptoms referable to this lesion were present.[1]

On the other hand numerous autopsies are on record in which a smaller lesion (softening, hemorrhage, etc.), placed a centimetre further back in the left frontal lobe, involving the posterior part of the third frontal gyrus or the band of white substance between it and the nucleus caudatus, has given rise to severe symptoms, hemiplegia or aphasia, singly or combined.

In the paper just quoted I have described such cases.

This case has the same negative importance as Case 4: serving to indicate the anterior limit of motor activities in the hemisphere. It shows that the lower part of the first and second frontal, and the orbital lobule of the frontal lobe, have no direct motor connections with peripheral

[1] A contribution to the study of localized cerebral lesions. Case 6. *Transactions of the American Neurological Association*, vol. ii, pp. 122–4, N. Y., 1877.

parts of the body; and, also, that these regions of the brain are non-excitable.

CONCLUSIONS.

The following conclusions may be legitimately drawn from the cases of localized cerebral disease (twelve in number) which I have published in the last four years:

1. The motor area of the cerebral cortex and allied white substance extends anteriorly as far as the lower half of the second and first frontal gyri, and posteriorly as far as the anterior part of the interparietal fissure. This statement is justified by Case 7 of my first paper (lesion of the left frontal lobe), and Cases 4 and 5 of the present paper.

2. The region lying between the limits indicated above, the middle regions of the hemisphere, on its convexity and (to a certain extent) on its median surface, including the posterior parts of the first and second, the whole of the third, frontal gyri, the whole of the ascending frontal and ascending parietal gyri, with their terminations in the longitudinal fissure known as the paracentral lobule, with probably the upper parietal lobe,—all these cortical parts, with their associated segments or fasciculi of white matter, have strong motor functions, being in direct relation with the muscles of the face, tongue, arm, and leg. This general statement is supported by the remaining nine cases in the two essays, in which destructive lesions of this area gave rise to spasm or paralysis on the opposite side of the body.

A further and more elaborate induction is permissible from these nine positive cases:

a. The lower part of the third frontal gyrus is intimately connected with the organs of speech (and the function of language).—Cases 1, 2, 3, and 4 of former essay.

b. The middle parts of the ascending frontal and ascending parietal gyri are directly connected with the arm of the

opposite side.—Case 5 of first essay, and Case 1 of present paper.

c. The upper or posterior part of the ascending frontal and ascending parietal gyri, and the paracentral lobule (also the upper parietal lobule?), are directly connected with the lower and upper extremities of the opposite side, and perhaps more closely with the leg.—Case 6 of first essay, Cases 2 and 3 of present paper.

I can not offer any case bearing on the questions of the location of the facial and ocular centres; though I now have under study a living case of exquisite epileptiform facial monospasm, which has been controlled by a strict bromide treatment.